

In the claims:

16. (presently amended) A method of inhibiting tumor growth in a mammal comprising administering to said mammal a compound which inhibits an enzymatic activity of HAAH, wherein said HAAH comprises the amino acid sequence of SEQ ID NO:2 and wherein said compound is a dominant negative mutant of said HAAH, said mutant comprising a mutation in a catalytic domain of HAAH, said catalytic domain comprising residues 650-700 of SEQ ID NO:2.

17. (canceled) The method of claim 16, wherein said enzymatic activity is hydroxylase activity.

18. (canceled) The method of claim 16, wherein said compound is a dominant negative mutant of HAAH.

19. (canceled) The method of claim 18, wherein said dominant negative mutant HAAH comprises a mutation in a catalytic domain of HAAH.

20. (canceled) The method of claim 16, wherein said compound is an HAAH-specific intrabody.

21. (canceled) The method of claim 16, wherein said compound is L-mimosine.

22. (canceled) The method of claim 16, wherein said compound is a hydroxypyridone.

39. (presently amended) ~~The method of claim 16~~ A method of inhibiting tumor growth in a mammal comprising administering to said mammal, wherein said compound is an antibody or fragment thereof which binds to HAAH an epitope within a catalytic domain of HAAH, said antibody being linked to a cytotoxic agent, wherein said domain comprises residues 650-700 of SEQ ID NO:2.

40. (canceled) The method of claim 39, wherein said antibody or fragment thereof binds to an epitope in the catalytic domain of HAAH.

41. (canceled) The method of claim 16, wherein said compound is a single chain Fv molecule.

42. (presently amended) The method of claim ~~16~~ 39, wherein said compound is a FB50 antibody.

43. (presently amended) The method of claim ~~16~~ 39, wherein said compound is a FB50 single chain Fv molecule.

44. (presently amended) The method of claim ~~19~~ 16, wherein said mutation comprises a substitution or deletion of a histidine residue in said catalytic domain of HAAH.

45. (canceled) The method of claim 19, wherein said mutation is located between residues 650-700 of SEQ ID NO:2.

46. (presently amended) The method of claim ~~19~~ 16, wherein said mutation is a substitution or deletion at residue 675 of SEQ ID NO:2.

47. (presently amended) The method of claim 19-A method of inhibiting tumor growth in a mammal comprising administering to said mammal a dominant negative mutant of HAAH, wherein said HAAH comprises the amino acid sequence of SEQ ID NO:2 and wherein said mutant comprises mutation is a substitution or deletion at residue 679 of SEQ ID NO:2.

48. (presently amended) The method of claim 19-A method of inhibiting tumor growth in a mammal comprising administering to said mammal a dominant negative mutant of HAAH, wherein said HAAH comprises the amino acid sequence of SEQ ID NO:2 and wherein said mutant comprises mutation is a substitution or deletion at residue 690 of SEQ ID NO:2.

49. (presently amended) The method of claim ~~19~~ 47 or 48, wherein said compound is administered directly into a tumor site.

50. (presently amended) The method of claim ~~19~~ 47 or 48, wherein said compound is administered systemically.

51. (previously amended) The method of claim 16, wherein said tumor is selected from the group consisting of colon cancer, breast cancer, pancreatic cancer, liver cancer, and cancer of the bile ducts.

52. (previously amended) The method of claim 16, wherein said tumor is a cancer of the central nervous system.

53. (previously amended) The method of claim 16, wherein said tumor is a hepatocellular

carcinoma.

54. (previously amended) The method of claim 16, wherein said tumor is a cholangiocarcinoma.

55. (previously amended) The method of claim 16, wherein said tumor is a glioblastoma.

56. (previously amended) The method of claim 16, wherein said tumor is a neuroblastoma.

57. (newly added) A method of inhibiting growth of a tumor in a mammal comprising administering to said mammal an antibody selected from the group consisting of 5C7 produced by hybridoma ATCC designation PTA 3383, 19B produced by hybridoma ATCC designation 3384, and 86A produced by hybridoma ATCC designation 3385.